

Carmelo Bosco · Marco Cardinale · Olga Tsarpela

## Influence of vibration on mechanical power and electromyogram activity in human arm flexor muscles

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**Abstract** The aim of this study was to evaluate the influence of vibration on the mechanical properties of arm flexors. A group of 12 international level boxers, all members of the Italian national team, voluntarily participated in the experiment: all were engaged in regular boxing training. At the beginning of the study they were tested whilst performing forearm flexion with an extra load equal to 5% of the subjects' body mass. Following this, one arm was given the experimental treatment (E; mechanical vibration) and the other was the control (no treatment). The E treatment consisted of five repetitions lasting 1-min each of mechanical vibration applied during arm flexion in isometric conditions with 1 min rest between them. Further tests were performed 5 min immediately after the treatment on both limbs. The results showed statistically significant enhancement of the average power in the arm treated with vibrations. The root mean square electromyogram (EMG<sub>rms</sub>) had not changed following the treatment but, when divided by mechanical power, (*P*) as an index of neural efficiency, it showed statistically significant increases. It was concluded that mechanical vibrations enhanced

muscle *P* and decreased the related EMG/*P* relationship in elite athletes. Moreover, the analysis of EMG<sub>rms</sub> recorded before the treatment and during the treatment itself showed an enormous increase in neural activity during vibration up to more than twice the baseline values. This would indicate that this type of treatment is able to stimulate the neuromuscular system more than other treatments used to improve neuromuscular properties.

**Key words** Vibrations · Mechanical Power

### Introduction

The influence of resistance exercise on the neuromuscular properties of human skeletal muscles has been studied over the years; as a result it has been observed that, in response to such a stimulus, changes within the muscle itself constitute the most important adaptation (Sale 1988; Behm 1995). Neural adaptations have been indicated as the first changes occurring in the muscle, permitting gains in muscle strength and power in the early stages of a resistance exercise programme in the absence of increases in cross-sectional area of the muscle (Behm 1995; Costill et al. 1979; Moritani and DeVries 1979). The first phase of adaptation has in fact been universally attributed to an improvement in neural factors, the myogenic factors becoming more important as the adaptations continue over several months (e.g. McDonagh and Davies 1984). Research conducted on the effects of resistance training have shown that specific adaptations occur depending upon the training programme employed (Sale and MacDougall 1981).

In this respect it is important to consider the importance of specificity of training in producing particular adaptations. When dealing with a sporting performance such as the punch in boxing the aim of a resistance training programme would need to be specific in improving the segmental velocities of the arm, increasing in this way the speed of this unloaded movement which is

C. Bosco · O. Tsarpela  
University of Rome-Tor Vergata,  
Fondazione "Don Gnocchi", Rome, Italy

C. Bosco  
Department of Biology of Physical Activity,  
University of Jyväskylä, Finland

C. Bosco  
Department of Biomechanics,  
Hungarian University of Physical Education,  
Budapest, Hungary

C. Bosco · M. Cardinale · O. Tsarpela  
Italian Boxing Federation

M. Cardinale  
C.T.O. Hospital, Spinal Unit, Rome, Italy

C. Bosco (✉)  
C/o Società Stampa Sportiva,  
Via G. Guinizzelli, 56,  
I-00152 Rome, Italy

the most important technical skill in boxing. Previous studies have shown that training with heavy loads, with low velocities of execution, primarily produces changes in the force part of the force-velocity curve, while training with light loads affects primarily the velocity part (Kaneko et al. 1983). The mechanisms underlying this effect of velocity specificity have not been clearly defined, most importance however has been given to neural adaptations such as improved coordination, increased activation of prime mover muscles (Moritani and DeVries 1979), recruitment and synchronization.

It has been suggested that muscle stimulation by vibration may induce improvements in the mechanical power of the lower limbs in elite athletes (Bosco et al. 1998) through a neural adaptation. Moreover, Burke et al. (1996) have shown that facilitation of the excitability of the patellar tendon reflex can be elicited through vibration applied to the quadriceps muscle. It has been shown that mechanical vibration (10–200 Hz) administered to tendons or muscles can cause a reflex response (Hagbarth and Eklund 1965). This particular reflex activity has been named the “tonic vibration reflex”. It is still controversial as to whether it can be elicited by low vibration treatment (30 Hz) or just from frequencies of about 100 Hz and an amplitude of 1 mm (Latash 1998), even if it has been suggested to occur during whole body vibration at frequencies ranging from 1 to 30 Hz (Seidel 1988).

The aim of this study was to analyse the effects of vibration on the neuromuscular behaviour of arm flexors in elite boxers and to gain knowledge from this to verify its possible use as a means of training for improving explosive arm movements through an improvement of neuromuscular efficiency.

## Method

### Subjects

A group of national level boxers (Italian national team) volunteered as subjects for the present study. All of them had been competing for several years and were participating regularly in boxing training programmes. Full advice was given to the volunteers regarding the possible risk and discomfort that might be

**Table 1** Reliability of two successive trials (trials 4, 5) of the average power ( $\bar{P}$ ), electromyogram (EMG) and  $EMG/\bar{P}$  measured during arm flexion executed with a load of 5% of subject's body mass, in the treatment and in the control arms ( $n = 24$ ), before vibration treatment.  $r$  Pearson product moment correlation coefficient,  $CV$  coefficient of variation for repeated measurements

| Variables                  |      | Trial 4 | Trial 5 | $r$   | $CV$ |
|----------------------------|------|---------|---------|-------|------|
| $\bar{P}$ (W)              | Mean | 61.1    | 59.9    | 0.96* | 5.4  |
|                            | SD   | 15.2    | 15.6    |       |      |
| EMG ( $\mu$ V)             | Mean | 375.0   | 356.7   | 0.91* | 15.0 |
|                            | SD   | 183.0   | 170.8   |       |      |
| $EMG/\bar{P}$ ( $\mu$ V/W) | Mean | 6.45    | 6.23    | 0.93* | 14.1 |
|                            | SD   | 3.55    | 3.93    |       |      |

\*  $P < 0.001$

involved and all the subjects gave their written informed consent to participate in the experiment, which had been approved by the Ethics Committee of the Italian Society of Sport Science. Subjects with a previous history of fractures or bone injuries were excluded from the study. None of the subjects smoked and no medication was being taken by the athletes which would have been expected to affect physical performance.

### Mechanical power measurements

Each subject was tested during forearm flexion with an extra load equal to 5% of their body mass ( $m_b$ ). All the subjects performed a maximal dynamic elbow flexion with each limb. Five attempts were made with 1-min intervals between each. Since two or three trials were needed to reach a plateau in performance, the last two trials of each set of measurements recorded from each limb were averaged and used for statistical analysis as has been recommended by Tornvall (1963) and Bosco et al. (1995). Both limbs were tested separately, with a 5-min interval between tests. After this evaluation, the arms were randomly assigned to receive the treatment (E) or to be the control (C). After the vibration treatment both limbs were retested (post) using the same procedure as before (pre). During each elbow flexion, the mechanical power ( $P$ ) was calculated, and electromyogram (EMG) activity was recorded from the biceps brachii muscle. The movement of the elbow flexion was monitored with a sensor (encoder) machine (MuscleLab–Bosco System, Ergotest Technology A.S., Langensund, Norway), interfaced to a personal computer (Fig. 1). When the loads were moved by the subjects, a signal was transmitted by the sensor at every 3 mm of displacement. Thus it was possible to calculate several parameters such as average velocity, acceleration, average force, and average power ( $\bar{P}$ ), corresponding to the load displacements (for details see Bosco et al. 1995). However, since it has been shown that  $\bar{P}$  is the most sensitive parameter among all the mechanical variables studied, only  $\bar{P}$  was considered for statistical analysis (Bosco et al. 1995).

### EMG analysis

The signals from the biceps brachii muscle in either the E or C groups were recorded with bipolar surface electrodes (interelectrode distance 1.2 cm) including an amplifier (gain 600, input impedance 2 G $\Omega$ , CMMR 100 dB, band-pass filter 6–1500 Hz; Biochip Grenoble, France) fixed longitudinally over the muscle belly. The MuscleLab converted the amplified raw EMG signal to an average root-mean-square (rms) signal via its built in hardware circuit network (frequency response 450 kHz, averaging constant 100 ms, total error  $\pm 0.5\%$ ). The  $EMG_{rms}$  was expressed as a function of the time (millivolts or microvolts). Since the  $EMG_{rms}$  signals were used in relation to bio-mechanical parameters measured using MuscleLab, they were simultaneously sampled at 100 Hz. The subjects wore a suit next to the skin to prevent the cables from swinging and from causing movement artefacts. A personal computer (PC 486 DX-33 MHz) was used to collect and store the data. The  $EMG_{rms}$  analysis was performed during forearm flexion and during the period of vibration.

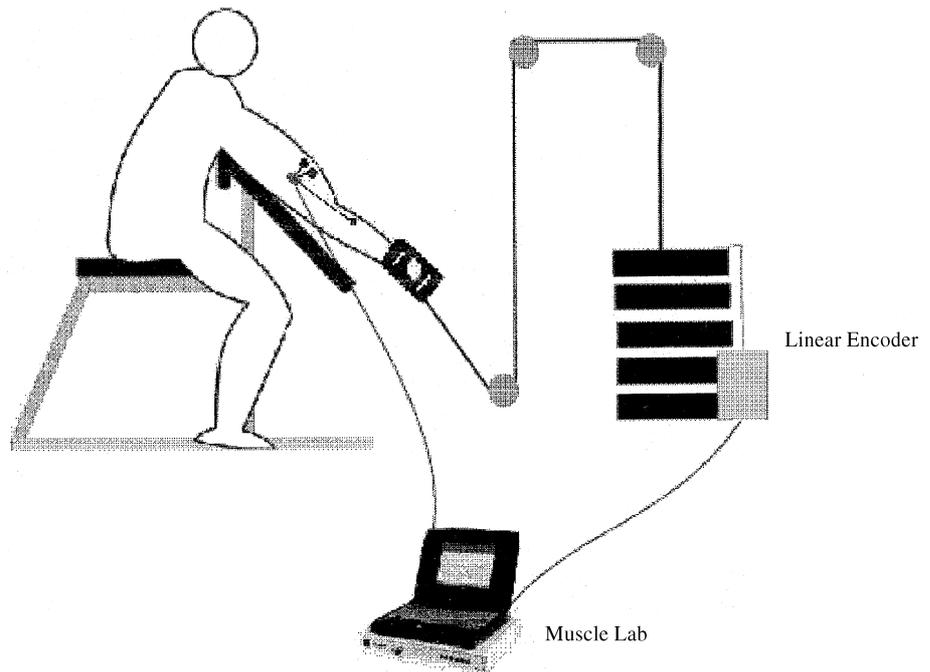
### Reliability of the measurements

Table 1 gives mean values, standard deviations, coefficients of correlation ( $r$ ) and coefficients of variation ( $CV$ ) of the last two trials (4, 5) used for statistical analysis. The  $CV$  showed results ranging from 6% to 15%, while the  $\pm$  found were between 0.73 and 0.93 ( $P < 0.001$ ).

### Treatment procedures

The subjects were exposed to a vibration treatment (VT) using the GALILEO 2000 device (Novotec, Pforzheim, Germany). In a

**Fig. 1** Test apparatus and subject's placement during the experiment



parallel experiment (Bosco et al. in press) it has been noted that during vibration at 30 Hz the EMG signal of the biceps brachii muscle reached its greatest activity, thus this frequency was chosen in the present study (displacement = 6 mm; acceleration =  $34 \text{ m} \cdot \text{s}^{-2}$ ). The subjects were exposed five times for durations of 60 s with 60 s of rest between each treatment

#### Type of treatment employed

The subjects were exposed to VT by gripping the vibrator machine in standing position. During VT, the arm was kept in semi-flexed position while gripping the machine (internal angle between forearm and arm was about 2.5 rad, and load was 2.8 kg). The arm exposed to VT was assigned as E, while the other not exposed was assigned as C. The arms randomly assigned as E or C demonstrated similar mechanical behaviour before VT (see Fig. 2). During the tests, administered pre and post the five VT, both limbs, assigned either to E or to C, lifted the same vibrating dumbbell.

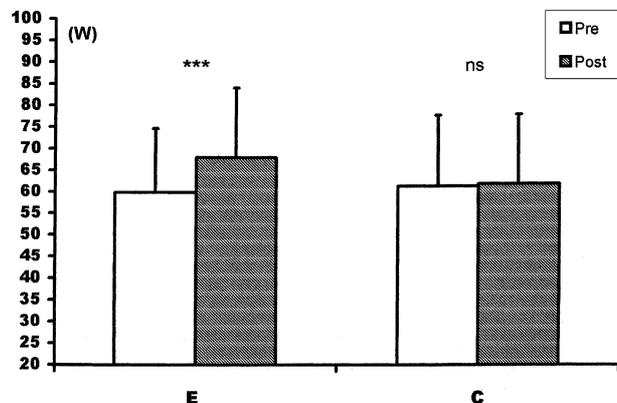
The subjects gripped the vibratory machine in standing position to avoid dispersion of the vibrations through contact with the special bench used for the tests where a particular design was necessary to ensure a standardised motion. Room temperature was kept constant at 22°C to avoid changes in EMG intensity when sitting or standing as has been suggested elsewhere (Meigal et al. 1996).

#### Statistical methods

The usual statistical methods were employed. The Pearson product moment correlation coefficient ( $r$ ) was used for test re-test measurement reliability. The CV of test re-test measurements was calculated using the following equation (Thorstensson 1976):

$$CV = \left( 200 \times \frac{SD}{\sqrt{2}} \right) \times (x_1 + x_2)^{-1} \quad (1)$$

where  $x_1$  and  $x_2$  are the mean average values of two successive measurements and SD is the standard deviation of the mean differences between test re-test measurements.



**Fig. 2** Average mechanical power (ordinate) recorded during arm flexion of the treatment (E) and control (C) arm before (Pre) and after the treatment period (Post). Asterisk denotes statistically significant differences ( $P < 0.001$ ) between the test performed before and after the treatment period

## Results

### Mechanical behaviour

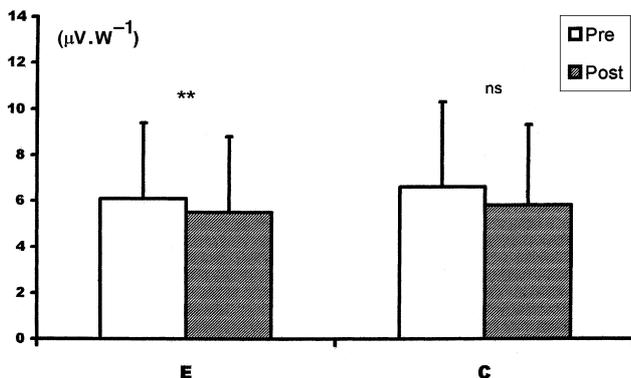
Before VT, the mechanical behaviour of both E and C demonstrated no statistically significant differences in  $\bar{P}$  calculated during elbow flexion with the extra load of 5% of  $m_b$ . After VT the arms of the E group showed a statistically significant improvement (pre vs post) for the  $\bar{P}$  ( $P < 0.001$ , Fig. 2). In contrast, the mechanical behaviour of the arms in C group, showed no changes in  $\bar{P}$  when the pre-post test analysis was performed (Fig. 2).

## Neuromuscular characteristics

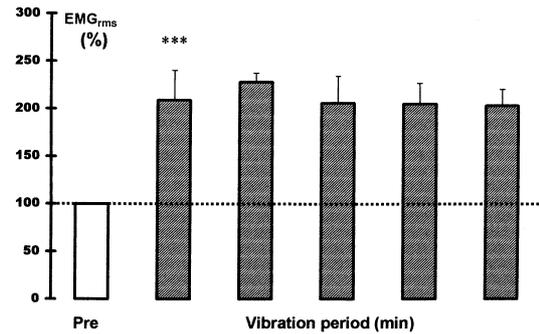
The  $EMG_{rms}$  detected in the biceps brachii muscles of E and C demonstrated no statistically significant differences in the pre-post test analysis. At the same time no differences were noted between the two treatment groups before and after VT. On the other hand, the  $EMG: \bar{P}$  ratio demonstrated in the E group a statistically significant decrement ( $P > 0.01$ ) after VT (Fig. 3). In the C group, the  $EMG: \bar{P}$  ratio after VT decreased, but statistical significance was not reached (Fig. 3). The  $EMG_{rms}$  collected from the biceps brachii muscles of E group before and during the VT, showed a significant enhancement during VT. The  $EMG_{rms}$  collected separately during each minute of VT are given as a percentage of the EMG found during rest recorded just before VT (Fig. 4). Statistically significant enhancement was observed when the EMG activity recorded during VT was compared to pre-vibration values ( $P < 0.001$ , Student's *t*-test, paired values), for all 5 min monitored. However, the analysis of variance employed to detect differences among means of the EMG collected during the experimental 5 separated min, showed no statistical differences.

## Discussion

As expected the pre compared to the post test analysis performed for the C group demonstrated no changes in the elbow flexor muscle *P*. These are not novel observations, since using a similar dynamometer Bosco et al. (1995) have found no changes, during a test re-test protocol, in the muscle *P* of athletes who throw. In contrast, in the present study, a significant increase in  $\bar{P}$  was noted in the elbow flexor muscles, following 5 min of VT (Fig. 2). Facilitation of the excitability of the spinal reflex has been elicited through vibration given to the quadriceps muscle (Burke et al. 1996). The possi-



**Fig. 3** Electromyogram/average power (ordinate) recorded during arm flexion of the treatment (E) and control (C) arm before (Pre) and after the treatment period (Post). Asterisks denote statistically significant differences ( $P < 0.01$ ) between the test performed before and after the treatment period



**Fig. 4** The electromyogram root-mean square  $EMG_{rms}$  given as a percentage of baseline (100%) recorded during each of the five repetitions lasting 1 min representing the treatment condition. The unfilled column is the baseline and filled columns are the treatment values. Asterisks denote statistically significant differences ( $P < 0.001$ ) between the baseline value and values recorded during the treatment

bility that vibration may elicit excitatory inflow through muscle spindle- $\alpha$ -motoneuron connections in the overall motoneuron inflow has been suggested also by Lebedev and Peliakov (1991). It has been demonstrated that vibration drives  $\alpha$ -motoneurons via a Ia neuron loop producing force without descending motor drive (Rothmuller and Cafarelli 1995).

Even if, as has been suggested, the vibration reflex, like the tendon jerk reflex, operates predominantly or exclusively via  $\alpha$ -motoneurons and does not use the same cortically originating efferent pathways, as has been shown to be the case when performing voluntary contractions (Burke et al. 1976), the possibility that VT can alter voluntary movement cannot be excluded. These suggestions are supported by the present findings. In fact the EMG recorded in the biceps brachii muscles of E showed a significant enhancement ( $P < 0.001$ ) of the neural activity during VT (Fig. 4), compared to normal.

It has been shown that vibration induced activation of muscle spindle receptors, not only in the muscle to which the vibration was applied, but also in the neighbouring muscles (Kasai et al. 1992). Mechanical vibration (10–200 Hz), applied to muscle belly or tendon, has been shown to elicit reflex contraction (Hagbarth and Eklund 1965). This response has been named “tonic vibration reflex” (TVR). It is not known whether it can be elicited by low vibration treatment (30 Hz), even though it has been suggested to occur during whole body vibration at frequencies ranging from 1 to 30 Hz (Seidel 1988).

In the present study the influence of skin mechanoreceptors, which would have activated some muscle receptors, has also been considered. This phenomenon would have been due to the fact that the vibrating dumbbell was gripped with the hand and at the frequency used in the experiment (30 Hz) the Meissner corpuscles located in the hand could possibly have triggered muscle spindle activation as has been indicated elsewhere (Hollins and Roy 1996). Previous findings have also

suggested that there is some dynamic fusimotor drive to completely relaxed muscles operating in the human hand, and that this drive is altered reflexly by cutaneous afferent input from the hand itself when trains of stimuli at non-noxious levels are applied to the palmar surface of the fingers (Gandevia et al. 1994).

The influence of Pacinian corpuscle has been discarded since the indications in the literature refer to their activation at higher frequencies of stimulation (Hamano et al. 1993; Hollins and Roy 1996). In a study conducted by Kodachi et al. (1987) a vibratory stimulus applied to the skin of the finger tip induced a flexion reflex in that finger and when the characteristics of the reflex were compared to those of TVR they showed wider spreading effects, the conclusion of the researcher being that this reflex could have originated from skin mechanoreceptors and involve a long loop. However, since the aim of the present study was not to investigate the influence of skin mechanoreceptors in reflex activation, we did not consider their influence since they were more likely to have influenced reflex activity of the hand rather than the biceps brachii muscle.

The improvement in muscle performance after VT has been quoted (Bosco et al. 1998) to be similar to that occurring after several weeks of heavy resistance training (e.g. Ikai and Fukunaga 1970; Coyle et al. 1981; Hakkinen and Komi 1985). In fact, the improvement in muscle function after resistance training has been attributed to enhancement of neuromuscular behaviour caused by an increasing activity of higher motor centres (Milner-Brown et al. 1975). The improvement of muscle performance induced by VT would suggest that a neural adaptation had occurred in response to VT. In this connection, the duration of the stimulus would seem to be important. Adaptive response of human skeletal muscle to simulated hypergravity conditions (1.1 *g*), applied for only 3 weeks, has been found to cause an improvement in the behaviour of the leg extensor muscle (Bosco 1985). Thus it is likely that both neural adaptation and length of the stimulus play important roles in the improvement of muscle performances (e.g. Bosco 1985).

During VT the elbow flexors were stimulated for a total of 300 s. This duration was similar to that required to flex the elbow 600 times with a load equal to 5% of the subject's  $m_b$ . Such a number of repetitions if made three times a week with 50 repetitions each time would take 1 month. The great initial increases noted in muscle strength during the early weeks of intense strength training has been explained through the increases in maximal neural activation (e.g. Moritani and De Vries 1979). It is not easy to explain how the increased neural output may occur or the intrinsic mechanism of neural adaptation. Furthermore, a net excitation of the prime mover motoneurons could result from increased excitatory input, reduced inhibitory input or both (e.g. Sale 1988). However, in the present experiment, the improvement of  $P$ , noted after VT, was not achieved by a parallel potentiation of the EMG activity recorded in the biceps brachii muscle (Table 2). Indeed after VT the

EMG activity in biceps brachii muscle was found to be rather low ( $P < 0.01$ ), even if during VT an increment of neural input to the muscle occurred (Fig. 4). In this respect the decrease of EMG activity of the biceps brachii muscle associated with increase of  $P$ , showed that VT induced an improvement of the neuromuscular efficiency of the elbow flexors.

It has been suggested that vibration leads to vasodilatation, attributable to a local axon reflex (Nakamura et al. 1996). In the present study it was most probable that VT could have induced vasodilatation and consequently increased the temperature of the biceps brachii muscle. It is difficult to explain the decrease of EMG activity through this effect, since the phenomenon has not yet been well explained, as has been demonstrated by the contrasting results in the international literature. In fact it has been shown that no changes occur in the relationship between static force and  $EMG_{rms}$  due to temperature changes (Holewijn and Heus 1992). On the other hand, a decrease of EMG activity has been reported to be associated with a reduction of temperature and muscle performance (e.g. Oksa et al. 1997). In contrast, increasing room temperature, has been shown to decrease the EMG/force relationship (Bell 1993). A reduction of EMG activity associated with a given level of force production has been shown to occur during the later training weeks of a long-term resistance training programme (Komi et al. 1978). On the other hand, when strength athletes have trained for few weeks with sub-maximal loads of 70%–80% of 1 repetition maximum the maximal EMG decreased (Hakkinen and Komi 1985). It has also been argued that in the presence of TVR the vibration induced suppression of motor output in maximal voluntary contractions probably does not depend on voluntary commands (Bongiovanni et al. 1990). It has been suggested that the contributing mechanism might be vibration induced presynaptic inhibition and/or transmitter depletion in the group Ia excitatory pathways which constitute the afferent link of the  $\gamma$ -loop (Bongiovanni et al. 1990). However, attribution of the lower EMG found after VT to the effect of TVR is rather difficult, since  $P$  was enhanced.

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